value in conjunction with Dr. Archibald from the bacteriological side as well as the clinical.

In the treatment of movable kidney the correction of the ptosis is of first importance. I have shown the necessity of correcting bladder sag in order to cure a cystitis and the principles involved here are similar. We have a certain amount of stasis and with this stasis the urine is altered and loses its supposed antiseptic action, so of prime importance is the giving of drainage and not until then can we expect results from vaccines.

The best method of correcting the ptosis is a debatable question at present. We have swung strongly from the kidney fixation, largely, I believe, because too many tried to cure movable kidneys associated with enteroptosis that way. The cases not being segregated, many a movable kidney was corrected when the pathology from which the patient suffered was situated elsewhere. Again, many cases recurred, accounted for by one of two causes: those enteroptotic cases where kidney fixation without support of the other organs could never be expected to stay; secondly, the neglect of the abdominal support so necessary to use until the kidney has had time to reform its own bed and to give the patient a proper carriage. If our patient can be fitted with a proper front lacing corset, we have the means of testing out our diagnosis as we have with a pessary in bladder ptosis; but that, of course, is only a palliative method. Against the regular kidney belt I wish to enter a protest, for no other abdominal contrivance has such a power for evil in developing congestion in the pelvic organs. The two-part surgical corset in a thin individual, I have seen produce kidney crises by pinching the ureter and in the same type of individual the hope of fitting any kind of support is rather forlorn.

Having proven the condition of the patient to be due to the kidney ptosis, the type of operation to be done opens fresh opportunity for study. I am inclined to the method of Longyear combined with the fixation of the capsule, for we probably correct a colon sag if present. If my contention that the presence of a bacterial growth in the urine proves the kidney ptosis to be the cause of the pathological symptoms is correct, I believe that in the properly segregated cases we shall be doing more and more kidney fixation and after operation fitting rational corsets to our patients.

My premises, then, are:

That normal urine is sterile.

That the greater number of chronic urinary tract infections are associated with a bladder or kidney ptosis or both.

That the "unilateral nephritis" is a condition of infection having as its origin a kidney sag.

That many movable kidneys are without pathological significance because the muscle tone is unimpaired.

That when this muscle tone becomes impaired we have urine stasis and infection.

That every movable kidney is a latent source of trouble.

That in the bacteriological examination of the urine we have a means of diagnosing the pathological "floating kidney."

That the degree of symptomatology depends on the kind of infection and the sensibility of the patient.

That being able to diagnose positively a pathological "floating kidney" we will consider more seriously the operative treatment and the type of operation, for at best the kidney support is only temporary and often impossible of proper application.

Discussion.

Dr. J. Henry Barbat, San Francisco: Just seven years ago I read a paper on enteroptosis, but did not bring up the subject of the bacteriological findings in the urine. At that time I spoke of Edebohl's operation of decapsulation of the kidneys for nephritis, and showed that the cases which were really cured were those in which the kidneys were not organically diseased, but merely disturbed functionally, as the result of displacement.

Regarding the lacing of corsets, it does not matter whether the corset is laced in front or in back, if it supports the abdominal organs and kidney as well. The great difficulty is to get a support which will act in every case. We are far from perfection as yet and must use judgment in selecting appropriate measures to fit each case. The best corset which I have found is the long, straight front one with a heavy front steel. When the corset is laced there must be at least one inch between the lacing steels, so as to maintain the tension. As soon as the lacing pulls the edges together at the bottom, the corset no longer offers any support, and must be taken up. The top should be loose enough to allow the hand to go down to the waist line.

CARBOHYDRATE CURES IN DIABETES.*

By E. SCHMOLL, M. D., San Francisco.

A few years ago when Naunyn wrote his classical treatise on diabetes, the treatment of this metabolic disturbance seemed to be established on a foundation equally supported by our theoretical conception of the nature of the disease as well as by practical experience. I remember that, at that time discussing the pathology of glycosuria, Prof. Alonzo Taylor and I agreed that it was the clearest chapter of the pathology of metabolism, and that the only point needing further investigation was the prevention of acidosis.

Few years have passed since. To-day everything that seemed to be established, is again under discussion. The points on which our theoretical conception is based, are the centre around which the battle of scientific argumentation is fought. No development in the history of medical science demonstrates more clearly that the theory can only be viewed as a temporary résumé of all the known facts, and that any new phenomenon which cannot

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be accounted for, has to lead to a complete change of our theoretical conception.

Few facts in therapy are better established than the improvement of the diabetic disturbances under treatment leading to a disappearances of glycosuria. The secondary disturbance, like loss of weight, tendency to infection, pruritis, etc., fade as soon as the sugar disappears from the urine.

In the immense majority of cases, the carbohydrate contents of the food are the determining factor in glucose excretion; in a minority consisting principally of the severe cases, the glycosuria varies with the amount of proteid given in the food. Restriction of carbohydrates in the first class of diabetics, cutting down of the proteid contents of the food in the second, leads generally to a disappearance of the glycosuria and a marked improvement in the patient's conditions. These facts established by Naunyn and his school, constituted the basis on which our treatment had been built up.

Occasionally, however, certain cases were observed in which diabetics seemed to derive benefit from the feeding of carbohydrates. Certain cures were advocated in which the principle factor seemed to consist of feeding large quantities of carbohydrates like milk, rice, potatoes and milk sugar. They appeared, however, so irrational that they rapidly fell into oblivion until V. Noorden published his paper on oatmeal cures, in which he described a number of well observed cases, favorably influenced by an exclusive carbohydrate diet. At first this communication was received with a great deal of scepticism, overcome, however, in the following years by the weight of concording evidence.

The treatment followed by V. Noorden required a preparatory series of two or three vegetable days to reduce the glycosuria and increase the ability of the diabetic to take care of ingested carbohydrates. This was followed up by two or three days, during which 200 grammes of oatmeal were given cooked with 200 grammes of butter with the occasional addition of a vegetable proteid or eggs. The oatmeal days were followed again by one or two vegetable days after which the classical strict diet was resumed.

The points upon which the success of the carbohydrate diet depended, were fomulated in the following way:

The oatmeal diet should never be given without the preceding vegetable days, as the diabetic organism has first to get rid of the accumulated surplus of carbohydrates. Nor should the return to the strict diet be made without the interpolation of a similar period. Particular stress was laid on the exclusion of all other carbohydrates during the oatmeal cure. Failure to observe this point very often produces a decrease of the tolerance and an increase of the ketonuria.

Animal proteid should be strictly excluded during the cure, as it annihilates the favorable action of an exclusive carbohydrate diet.

The result of the treatment will be more favorable the less proteid is given during the whole duration of the dietary change, so that in late years the results have become more uniform through the exclusion of all proteid addition.

The oatmeal period should never be continued longer than three or four days, as the favorable influence is superseded by a decrease in tolerance.

The advantages which V. Noorden claims for his oatmeal treatments may be classed under the following headings:

First: The action of oatmeal is specific and is either due to a different constitution and different resorption of the oatmeal starch, or to some ferments contained in the oatmeal which render utilization of oatmeal starch possible for the diabetic.

Second: The cure can only be applied in cases of severe diabetics, as light cases do not react to the oatmeal treatment. Amongst severe cases we find a number in which no favorable result can be observed after such treatment.

In a great majority of cases an increase of tolerance to carbohydrates is obtained. The favorable result is maintained during the following days of strict diet, so that the patient who could not be made sugar free on a strict diet, can be kept on the same diet without excreting any sugar.

As a result of the increase of tolerance the ketonuria very often disappears or is diminished to such a degree that an imminent coma may be averted.

It will be my endeavor to discuss the different claims of V. Noorden by analyzing the results in a great number of well-observed cases of diabetes.

First specificity of the oatmeal.

The specific action of oatmeal was at first accepted and all the following investigations were carried out with it, notwithstanding the results recorded by the old writers by means of different carbohydrates.

A great deal of work was done to find out whether the starch of the oatmeal could be utilized on account of a different chemical constitution, or whether some ferment-like substance influenced the utilization of the carbohydrates. Klotz, Lang and Magnus-Levy were able to show that bacterial decomposition of oatmeal starch as well as its resorption differed quite a good deal from other starches.

All these investigations, however, were made useless by the discovery that a number of different carbohydrates, if fed under the same precautions as oatmeal, led to an increase in tolerance.

Schmidt and his pupils, especially L. Blum demonstrated that very similar results could be obtained with other carbohydrates, provided the cure was carried out according to the original method of V. Noorden.

A number of cases treated at different periods with oatmeal, and afterwards with corresponding amounts of flour allow a comparison.

S.—31 years of age; male. A severe case of diabetes. Entered treatment 1910. The first oatmeal cure was undertaken in January, 1911.

Date, January 17; Total Amount, 2200; Specific Gravity, 1031; Reaction, Alkal. Intake—N., 8.34; Fat, 252;

C. H., 34; Cal., 2611. Output—Sugar, per cent., 2.2; Acetone, +; Diacetic Acid, ++; Ammonia, 0.2. Diet and Remarks—Proteid Fat.

Date, January 18; Total Amount, 1700; Specific Gravity, 1026; Reaction, Alkal. Intake—N., 11.02; Fat, 301; C. H., 170; Cal., 3783. Output—Sugar, Negative; Acetone —; Diacetic Acid, ++; Ammonia, 1.3. Diet and Remarks—Oatmeal and Eggs.

Date, January 19; Total Amount, 1900; Reaction, Alkal. Intake—N., 11.2; Fat, 301; C. H., 170; Cal., 3783. Output—Sugar, Negative; Acetone, +; Diacetic Acid, +; Ammonia, 0.1. Diet and Remarks—Oatmeal and Eggs.

Date, January 20; Total Amount, 2300; Specific Gravity, 1020; Reaction, Acid. Intake, N. 11.2; Fat, 285; C. H., 170; Cal. Net, 3555. Output—Sugar, Negative; Acetone, ++; Diacetic Acid, ++; Ammonia, 0.3. Diet and Remarks—Oatmeal and Eggs.

Date, January 21; Total Amount, 2300; Specific Gravity, 1019; Reaction, Alkal. Intake—N., 11.2; Fat, 287; C. H., 170; Cal. Net, 3571. Output—Sugar, per cent., 4.3; Acetone, +; Diacetic Acid, ++; Ammonia, 0.2. Diet and Remarks—Oatmeal and Eggs.

Date, January 22; Total Amount, 2450; Specific Gravity, 1020; Reaction, Acid. Intake—N., 4.79; Fat, 102; C. H., 67; Cal., 1390. Output—Sugar, Negative; Acetone, +; Diacetic Acid, ++; Ammonia, 0.2. Diet and Remarks—Vegetables and Eggs.

Date, January 23; Total Amount, 2050; Specific Gravity, 1020; Reaction, Alkal. Intake—N., 4.23; Fat, 118; C. H., 44; Cal., 1415. Output—Sugar, negative; Acetone, ++; Diacetic Acid, ++; Ammonia, 0.17. Diet and Remarks—Vegetables and Eggs.

During the oatmeal period the glycosuria disappeared immediately on the first day, to return on the third and to increase on the fourth day, showing the disadvantage of a long-continued carbohydrate diet. Notwithstanding this mistake the patient on return to the strict proteid fat diet remained sugar-free.

The patient returned in 1913, in a very much emaciated condition and with a very lowered tolerance. On entering the hospital he showed some premonitory symptoms of coma. He was put on a vegetable diet followed by flour diet.

Date, January 8, 1913; Total Amount, 3100; Specific Gravity, 1029; Reaction, Acid. Intake—N., 3.1; Fat, 168; C. H., 111; Cal., 2167; Cal. Net, 2043. Output—Sugar, Per Cent., 1.0, Total, 31; Acetone, +++; Diacetic Acid, +++; Ammonia, 4.3. Weight—Gain, 2¼. Diet and Remarks—Vegetables.

Date, January 9, 1913; Total Amount, 3000; Specific Gravity, 1031; Reaction, Acid. Intake—N., 3.0; Fat, 222; C. H., 187; Cal., 2833; Cal. Net, 2499. Output—Sugar, Per Cent., 2.7, Total, 81; Diacetic Acid, +++; Ammonia, 3.6. Weight—Gain, 1¾. Diet and Remarks—Flour and Whisky.

Date, January 10, 1913; Total Amount, 2650; Specific Gravity, 1032; Reaction Slight Acid. Intake—N., 3.0; Fat, 222; C. H., 187; Cal., 2823; Cal. Net, 2495. Output—Sugar, Per Cent., 3.1, Total, 75; Acetone, +; Diacetic Acid, +++; Ammonia, 1.3. Weight—Gain, 3½. Diet and Remarks—Flour and Whisky.

Date, January 11, 1913; Total Amount, 2200; Specific Gravity, 1031; Reaction, Neutral. Intake—N., 3.0; Fat, 222; C. H., 187; Cal., 2823; Cal. Net, 2543. Output—Sugar, Per Cent., 3.2, Total, 68; Acetone, ++; Diacetic Acid, ++++; Ammonia, 1.7. Weight—Gain, 3. Diet and Remarks—Flour and Whisky.

Date, January 12, 1913; Total Amount, 2350; Specific Gravity, 1028; Reaction, Acid. Intake—N., 4.0; Fat, 227; C. H., 110; Cal., 2861; Cal. Net, 2779. Output—Sugar, Per Cent., 0.8, Total, 19; Acetone, +; Diacetic Acid, ++++; Ammonia, 1.7. Weight—Gain, 3½. Diet and Remarks—Vegetables.

During the flour period the result was not nearly as good as under the oatmeal. The glycosuria failed to disappear, and the acidosis still showed itself in a very strong ferrichlorid reaction. Still the beneficial effects are apparent enough; he utilized over 100 grams of carbohydrates and the ammonia output, the most accurate index of acidosis, fell from 4.3 to 1.7 grs. in 24 hours. I would like to attract your attention to the phenomenal gain during the flour diet, largely due to the retention of water.

A better result was obtained in the case of Mr. McK., 28 years of age. Intermediary case.

Date, January 2; Total Amount, 4000; Specific Gravity, 1038; Reaction, Acid. Intake—N., 22.4; Fat, 140; C. H., 224; Cal., 2786; Cal. Net, 1634. Output—Sugar, Per Cent., 7.2, Total, 228; Acetone, —; Diacetic Acid, —; Diet and Remarks—Proteid Fat.

Date, January 3; Total Amount, 1500; Specific Gravity, 1027; Reaction, Acid. Intake—N., 7.8; Fat, 70; C. H., 64; Cal., 1530; Cal. Net, 1416. Output—Sugar, Per Cent., 1.9, Total, 28.5; Acetone, —; Diacetic Acid, —. Diet and Remarks—Vegetables.

Date, January 4; Total Amount, 2700; Specific Gravity, 1018; Reaction, Acid. Intake—N., 3; Fat, 212; C. H., 147; Cal., 2663; Cal. Net, 2519. Output—Sugar, Per Cent., 2.2, Total, 59.4; Diacetic Acid, —. Diet and Remarks—Flour.

Date, January 5; Total Amount, 1800; Specific Gravity, 1019; Reaction, Acid. Intake—N., 3; Fat, 222; C. H., 147; Cal., 2663; Cal. Net., 2519. Output—Sugar, Per Cent., 2, Total, 36; Acetone, —; Diacetic Acid, —. Diet and Remarks—Flour.

Date, January 6; Total Amount, 1200; Specific Gravity, 1018; Reaction, Acid. Intake—N., 4.4; Fat, 130; C. H., 89; Cal., 1613. Output—Sugar, Per Cent., 0.1, Total, 1.2; Acetone, Trace; Diacetic Acid, —. Diet and Remarks—Vegetables.

The sugar excretion fell under the influence of the exclusive flour diet to traces, which finally disappeared under the influence of a strict proteid-fat diet.

These observations prove fully that flour works very similarly to the oatmeal; its anti-ketonic action, however, does not seem to equal the influence of oatmeal. In a series of observations I have failed to obtain a disappearance of sugar; for these reasons I have given up the exclusive flour diet and returned to the oatmeal cure as the most efficacious form of the carbohydrate treatment.

Secondly: V. Noorden stated that the oatmeal cure could only be applied in severe cases, while light cases failed to respond to it.

It can be easily shown that this view cannot be substantiated. Amongst a great number of light cases treated by either the oatmeal or flour diet with equally satisfactory results, I shall cite the following:

Mrs. S., 62 years of age. Light case.

Date, February 25; Total Amount, 1400; Specific Gravity, 1030; Reaction, Acid. Intake—N., 2.3; Fat, 168; C. H., 43; Cal., 1965; Cal. Net, 1837. Output—Sugar, Per Cent., 2.3, Total 32; Acetone, —; Diacetic Acid, —. Diet and Remarks—Vegetables.

Date, February 26; Total Amount, 1450; Specific Gravity, 1021; Reaction, Acid. Intake—N., 3.8; Fat, 193; C. H., 76; Cal., 2273. Output—N., 7.3; Sugar, Large Trace; Acetone, —; Diacetic Acid, —; Ammonia, 0.7. Weight—Gain, ¾. Diet and Remarks—Vegetables.

Date, February 27; Total Amount, 1250; Specific Gravity, 1021; Reaction, Acid. Intake—N., 4.7; Fat, 183; C. H., 164; Cal., 2657; Cal. Net, 2578. Output—N., 5; Sugar, Per Cent., 1.5, Total, 19.8; Acetone, —; Diacetic Acid, —; Ammonia, 0.6. Weight—Loss, 1. Diet and Remarks—Flour.

Date, February 28; Total Amount, 1100; Specific Gravity, 1026; Reaction, Acid. Intake—N., 4.7; Fat, 183; C. H., 164; Cal., 2666; Cal. Net. 2566. Output—N., 5.3; Sugar, Per Cent., 2.3, Total, 25; Acetone, —; Diacetic Acid, —; Ammonia, 0.7. Weight—Loss, 1½. Diet and Remarks—Flour.

Date, March 1; Total Amount, 1200; Specific Gravity, 1024; Reaction, Acid. Intake—N., 3.4; Fat, 214; C. H., 53; Cal., 2163. Output—N., 5.6; Sugar, Trace; Acetone, +; Diacetic Acid, —; Ammonia, 0.7. Weight—Gain, 2. Diet and Remarks—Vegetables.

Date, March 2; Total Amount, 1950; Specific Gravity, 1017; Reaction, Acid. Intake—N., 14.2; Fat, 150; C. H., 47; Cal., 2649. Output—N., 7.4; Sugar, Trace; Acetone, +; Diacetic Acid, —; Ammonia, 0.8. Weight—Loss, 1. Diet and Remarks—Proteid, Fat.

Date, March 3; Total Amount, 1300; Specific Gravity, 1023; Reaction, Acid. Intake—N., 11.7; Fat, 216; C. H., 27; Cal., 2596. Output—N., 9.9; Sugar, Negative; Acetone, +; Diacetic Acid, —; Ammonia, 0.7. Diet and Remarks, Proteid, Fat.

In a number of cases this short cut may be used to render the patient sugar-free and the method may prove useful in the hands of the practitioner, who has no hospital facilities. As a general rule the old method of treatment by the carbohydrate free diet must be considered superior on account of its educational value.

After all the carbohydrate cure can be followed out only for a few days, while the proteid fat diet represents the standard the diabetic has to follow for the rest of his existence.

Thirdly: The increase of tolerance claimed by V. Noorden can be observed in a great many cases. A typical action was obtained in the following case:

S., 44 years of age. Severe case.

Date, October 11; Total Amount, 2600; Specific Gravity, 1027; Reaction, Acid. Intake—N., 23.2; C. H., 78.7; Cal., 4344; Cal. Net, 4288. Output—Sugar, Per Cent., 1.5, Total, 39; Acetone, +; Diacetic Acid, ++; Ammonia, 2.7. Diet and Remarks—Proteid, Fat.

Date, October 12; Total Amount, 1920; Specific Gravity, 1029; Reaction, Acid. Intake—N., 22.9; C. H., 82; Cal., 3586; Cal. Net, 3534. Output—Sugar, 0.7, Total, 13.4; Acetone, Trace; Diacetic Acid, ++; Ammonia, 1.8. Diet and Remarks—Proteid, Fat.

Date, October 13; Total Amount, 1290; Specific Gravity, 1028; Reaction, Acid. Intake—N., 11.2; C. H., 170.5; Cal., 3783. Output—Sugar, Trace; Acetone, Trace; Diacetic Acid, ++; Ammonia, 1.5. Diet and Remarks—Oatmeal.

Date, October 14; Total Amount, 980; Specific Gravity, 1029; Reaction, Acid. Intake—N., 10.6; C. H., 163.8; Cal., 1308; Cal. Net, 1212. Output—Sugar, Per Cent., 2.5, Total, 45; Acetone, Trace; Diacetic Acid, +++; Ammonia, 1.3. Diet and Remarks—Oatmeal.

Date, October 15; Total Amount, 1440; Specific Gravity, 1030; Reaction, Acid. Intake—N., 11.2; C. H., 170.5; Cal., 3785; Cal. Net, 3653. Output—Sugar, Per Cent., 2.3, Total, 33; Acetone, —; Diacetic Acid, +++. Diet and Remarks—Oatmeal.

Date, October 16; Total Amount, 2220; Specific Gravity, 1022; Reaction, Alkal. Intake—N., 23.1; C. H., 90.1; Cal., 3460. Output—Sugar, .02, Total, 4.4; Acetone, +++; Diacetic Acid, ++; Ammonia, 1.1. Diet and Remarks—Proteid, Fat.

Date, October 17; Total Amount, 1920; Specific Gravity, 1016; Reaction, Alkal. Intake—N., 22; C. H., 45.1; Cal., 3622. Output—Sugar, —; Acetone, —; Diacetic Acid, ++; Ammonia, 0.8. Diet and Remarks—Proteid, Fat.

Date, October 18; Total Amount, 2190; Specific Gravity, 1020; Reaction, Neutral. Intake—N., 13.8; C. H., 77.6; Cal., 2945. Output—Sugar, Trace; Acetone, Trace; Diacetic Acid, ++; Ammonia, 0.7. Diet and Remarks—Vegetables.

The patient who could not be made sugar-free on strict diet, failed to show any trace after a period of three oatmeal days.

The failure of the carbohydrate cure has become less and less frequent, since we have come to realize the importance of the restriction in proteid.

Formerly we used to give a vegetable proteid or eggs with the oatmeal diet, but results have been very much more uniform since the proteid contents of the food have been cut down.

Fourthly: The influence of the ketonuria is apparent in practically every case. Even in cases where no other favorable result can be obtained, the restriction in the acid excretion is very marked.

All the cases in which I have applied the carbohydrate cure, the influence of the acidosis has been most beneficial. I have seen a number of cases on the verge of diabetic coma in which the fatal issue has been delayed.

Not all the cases of acidosis call for the application of the carbohydrate cure. A few remarks about the physiology of ketonuria will help us to establish the correct indications.

The acetone bodies are normal intermediary products in the decomposition of fatty acid. Under normal conditions they are burned up, whenever carbohydrates are oxidized; as Rosenbach expresses it, they are burned up in the fire of the carbohydrates. Whenever carbohydrates are excluded from the diet acetone bodies begin to appear in the urine; and acidosis sets in. After a few days of the carbohydrate free diet, the acetone bodies begin to diminish and finally disappear. Evidently another substance, probably the proteid sugar, has taken the place of the ingested carbohydrates.

The acetonuria in diabetes therefore may be caused by two conditions. In the severe cases the diabetic has lost the ability to use sugar given under ordinary conditions, and his ketonuria is due to the complete absence of carbohydrates in the metabolic processes. In the light cases a certain percentage of the sugar is oxidized and therefore burns up the acetone bodies; if such a case is put on a carbohydrate free diet a physiological ketonuria follows.

Acetone, ferrichloride reaction and high ammonia output do not necessarily call for a carbohydrate cure; in each case the reason for the ketonuria has to be analyzed and the treatment shaped accordingly.

In cases where the ketonuria is due to the sudden change to the carbohydrate free diet, the increase in tolerance obtained soon allows the proteid sugar to take care of the acidosis. After a few days of strict diet the signs of acidosis disappear.

If the acidosis is due to the intensity of the diabetic disturbance, and whenever the conditions are given for the development of a diabetic coma, the institution of a carbohydrate cure becomes imperative. A strict proteid fat diet in such a case would easily lead to a coma; under the oatmeal diet a certain percentage of the starch is utilized and restricts the amount of acetone bodies entering circulation.

The carbohydrate cure should be followed as long as an increase in tolerance can be demonstrated. Very frequently, however, the sugar excretion begins to go up on the third or fourth day and with it the acidosis increases.

In such cases a return to the strict proteid fat diet is indicated; if a decided gain in tolerance can be obtained the acidosis soon diminishes and may finally disappear altogether.

A lasting improvement of the acidosis can only be expected from a strict proteid fat diet; a carbohydrate cure which necessarily fails after a few days, can only be considered as a momentary relief, in the most favorable case as a method leading to a better tolerance of the carbohydrate free diet. Our endeavor must be to always return to the strict proteid fat diet as soon as possible; the favorable result in cases with intense acidosis obtained by such treatment may be seen from the following table:

Date, October 11; Total Amount, 2600; Specific Gravity, 1027; Reaction, Acid. Intake—N., 23.2; C. H., 78.7; Cal., 4344; Cal. Net, 4288. Output—Sugar, Per Cent, 1.5, Total, 39; Acetone, +; Diacetic Acid, ++; Ammonia, 2.7. Diet and Remarks—Proteid and Fat.

Date, October 15; Total Amount, 1440; Specific Gravity, 1030; Reaction, Acid. Intake—N., 11.2; C. H., 170.5; Cal., 3785; Cal. Net, 3653. Output—Sugar, 2.3, Total, 33.1; Acetone, —; Diacetic Acid, +++. Diet and Remarks—Oatmeal.

Date, October 16; Total Amount, 2220; Specific Gravity, 1022; Reaction, Alkal. Intake—N., 23.1; C. H., 90.1; Cal., 3460. Output—Sugar, Per Cent., 0.2, Total, 4.4; Acetone, +++; Diacetic Acid, ++; Ammonia, 1.1. Diet and Remarks—Proteid and Fat.

Date, October 24; Total Amount, 2100; Specific Gravity, 1023; Reaction, Alkal. Intake—N., 11; C. H., 110; Cal., 2644. Output—Sugar, Trace; Acetone, —; Diacetic Acid, ++; Ammonia, 0.5. Diet and Remarks—Proteid and Fat.

Date, December 18; Total Amount, 2160; Specific Gravity, 1025; Reaction, Alkal. Intake—N., 16; C. H., 108; Cal., 3173. Output—Sugar, Very slight trace; Acetone, —; Diacetic Acid, —. Diet and Remarks—Proteid and Fat.

The limitations of the carbohydrate cure are drawn by its ability to increase tolerance. It should never be prolonged as soon as the glucose excretion increases.

In a great majority of cases this point is reached

within 3 or 4 days; other cases already described by Naunyn, as diabetes with paradoxical tolerance seem to stand it for a long time. In one case, where I combined the oatmeal with a vegetable diet, the tolerance kept increasing during a period of 3 weeks.

The reluctance with which the carbohydrate treatment was received by the medical profession was principally due to our inability to bring its results in accordance with our theoretical conception of diabetes.

Two theories have been advanced to explain the origin of diabetes. V. Noorden and his school have tried to find its origin in an increased production of sugar exceeding its assimilation and transformation into glycogen. On the other hand Naunyn and his pupils in accordance with the great majority of writers on the subject agreed that the glycosuria was due to the inability of the body tissues to oxidize sugar.

The crucical experiment which to my mind decides the question in favor of Naunyn's view was done by Starling. He transfused a normal heart and the heart of a dog, rendered diabetic by extirpation of the pancreas, with a solution containing glucose. While the normal heart performed its work at the expense of the glucose the diabetic heart failed to oxidize the sugar, showing that the diabetic tissues were unable to take care of the glucose.

During the oatmeal diet large amounts of carbohydrates are poured into the circulation and under their influence the body suddenly seems to regain its power to oxidize glucose.

Numerous attempts to explain this fact from the standpoint of the old established theory have failed. It was assumed that oatmeal starch was split up in the intestines beyond the glucose molecule, and that the products of fermentation so formed were accessible to the diabetic metabolism and still able to exert the influence of the intact molecule on the acetone bodies. All these attempts have failed and it was definitely established that the action of the oatmeal was not specific, and that the effect depended entirely on the form in which the starch was given.

The diabetic organizm therefore is able to take care of carbohydrates provided proteid consumption is kept down to the lowest possible level. In accordance with that we find that the carbohydrates cures are only successful as long as nitrogen excretion can be kept down to a low level of about 5 gramms per day. As soon as the nitrogen excretion increases the glycosuria goes up.

A number of facts are in accordance with the view that under certain conditions the diabetic organism can take care of sugar. Diabetic frogs survive the extirpation of the liver for a number of days, and during that time they fail to excrete any glucose in the urine. Geese after extirpation of the pancreas develop diabetes which, while typical in all its manifestations, shows an increase of glucose in the blood while the urine does not contain any sugar.

It seems to be established that under normal conditions an intimate relation exists between oxidation of proteids and carbohydrates. It is probably a disturbance in this relation which will be made to account for the inability of the diabetic to oxidize glucose.

To discuss this any further would lead us into the domain of theoretical speculation. What I wanted to do to-day was to bring a number of facts before you to demonstrate what a valuable asset we possess in the carbohydrate cure, and to show in which cases and under what conditions it could be used to advantage.

THE EFFECT OF A MOMENTARY CONTACT WITH AN 18,000 VOLT CURRENT.

By PHILIP KING BROWN, M. D., San Francisco.

C. S. Y. Age 30. F. H. negative. Freight conductor on S. P. R. R. Past history and discussion of similar accidents, negative. Always strong. Tub. glands in 1. neck removed 12 years ago but never inconvenienced him.

Present trouble. On Jan. 7th at 2 p. m. he was standing on iron rachet platform at one end of freight car 2 feet below top of car. The train was moving about six miles an hour, and the patient was holding on to the brake-wheel, his hands covered with extra heavy Napatan leather mitts without rivets. The patient knows nothing of what happened and recalls now nothing of pain or sensation of any kind except a momentary hissing, sputtering noise. Later he learned that he was struck in passing by a live electric wire carrying 18,000 volts. The contact was necessarily instantaneous. The wire was not broken, but patient says he understands it was temporarily suspended where it was. He evidently fell to the ground but was unconscious of anything, and does not remember losing consciousness or the fall in any way, although he fell nearly 30 feet. Four hours later he began to be conscious of surroundings, and remembers the pain of a needle used to suture the left ear which was badly torn in the fall.

The patient must have fallen free of the moving train, but no one saw him fall and he does not know where he struck. The only evidences of bruises were the torn ear, soreness of left shoulder, skin off right elbow and two knuckles of left hand and over left ulnar prominence. Besides this there were circular burns an inch in diameter at the hair line on left frontal region, on the plantar surface of left big toe, a smaller burn on the under surface of the adjoining toe at the tip and still others at the base of the little toe of same foot on the outer side and on the plantar surface of the foot two inches from burn on little toe. The woolen cap, shoe and woolen sock were similarly burned. The use of the left arm and leg was impaired but patient thought it due to fall and did not mention it for two days. The arm and leg felt sore and heavy and he had to be assisted in getting his clothes on. The hand was all right but shoulder could not be lifted or hand flexed and extended. The leg was less affected and when he walked slowly it seemed all right. In hurrying or climbing stairs it feels heavy and is moved slowly.

Improvement in use of left arm and leg has been very slight so that present condition May 1st